Research Paper | Medical Science | E-ISSN No : 2454-9916 | Volume : 3 | Issue : 7 | July 2017

ALTERATIONS IN ENZYME PARAMETERS IN SCORPION HETEROMETROUS FULVIPES WITH EXPOSURE TO SELECTED HEAVY METALS: MERCURY AND LEAD

M. V Raghavendra Rao [1] | Yogesh Acharya [1] | Samir Fatteh [1] | Sireeshabala A [1] | Amin Fateh [1] | Simi Paramban [1] | Jithender Kumar Naik [2] | Anush C Pawar [2] | Jayalakshmi G [3]

[1] Avalon University School of Medicine, Willemstad, Curacao.
[2] Osmania University, Hyderabad, Telangana, India.
[3] Gandhi Medical College, Hyderabad, India.

ABSTRACT

Heavy metal exposures in animals have various effects in metabolism of with alteration in biochemical constituents and enzyme levels. It is necessary that we documented heavy metals toxicities and take an appropriate precaution in mother and fetus to decrease its detrimental effects. An experimental study was performed with viviparous animal Heterometrous fulvipes to access the cumulative effect of chronic heavy metals, mercury and lead, exposure on the activity levels of the enzymes succinate dehydrogenase (SDH) and glutamate dehydrogenase (GDH). Chronic heavy metal exposure resulted in variation in the enzymes levels with increase in SDH and decreases in GDH in animal models, prompting a further study to consolidate the finding in human study.

KEY WORDS: Metals, Heavy, Toxicity, Animal models, Enzymes, Activity.

INTRODUCTION:

Heavy metals are believed to exert their influence on the activity of the enzymes playing a vital role in the biochemical transactions of a living system. Embryonic development is characterized by growth and formation of new tissues. The alterations in the activity of enzymes and/or embryonic tissues would invariably influence the developmental processes in viviparous animals as embryonic nourishment is provided by the maternal sources. The metabolic levels of the embryo can be expected to be different from those of the maternal animal. But the changes in the enzymatic activity causes shift in the metabolism of the maternal and embryonic tissue. This is influenced by heavy metals, which can be reflected in the form of deviations from the normal growth and development.

Heavy metals, like mercury and lead have different actions on different enzymes and their activity in animals. Exposure of the clam, *Katelysia opima* to mercuric chloride, elevated the activity of aminotransferase and reduced Na^+-K^+ ATPase activity (1). Exposure of *Heteropneustes fossilis* to a sublethal concentration of mercuric chloride inhibited the activity of alkaline phosphatase (AP) but elevated the activity of succinic dehydrogenase (SDH), pyruvate dehydrogenase (PDH) and cholinesterase in the brain tissue (2,3). The activity of hexokinase in intestine, kidney, and liver was decreased in fishes exposed to mercury. Similarly PDH, SDH, lactate dehydrogenase (LDH) and Malate dehydrogenase (MDH) were also significantly decreased. (4)

Toxicity as a result of heavy metals may adversely affect the aquatic flora and fauna. Growth parameters of fish may serve as important bio indicators against pollution (5). Sastry and Sukla (6) studied decreased activity of Glucose -6-phosphatase, hexokinase, LDH, SDH and MDH in liver and muscles of *Channa punctatus* after acute and chronic exposures to Cadmium for 4 months. Sakoori et al (7) reported the increase in LDH, which catalyzes the inter conversion of lactate and pyruvate depending on the availability of NAD (Coenzyme). Santana and Azariah (8) noticed disturbance in enzyme activities of the crab *Sesarma quadratum* after exposing to two sub lethal concentrations of copper chloride for 21 days. LDH activity was significantly elevated in muscle and hepatopancreas tissues where as SDH was suppressed in the crab muscle, gills and hepatopancreas.

Heavy metal stress is known to elevate the levels of Glutamate dehydrogenase (GDH) and separate aminotransferases (9). Heavy metal may cause injury to the organisms and the damaged tissues subsequently causing malfunction (10). Continuous exposure to sub lethal cadmium concentration resulted in significantly elevated levels of both aspartate aminotransferase (AST) and alanine aminotransferase (ALT) activity in *Oreochromis nilloticus* exposed cadmium (11). The sub lethal doses of mercury and lead administered to the gravid females every month from August to April depressed the SDH activity in the maternal hepato pancreas and pedipalpal muscle and the embryos, depression being statistically significant only in the case of lead. The decline in SDH activity was considered to reflect the suppression of oxidative metabolism by the heavy metals. (12)

The objective of this study is to determine the effect of heavy metals in the activity levels of SDH and GDH in viviparous animal *Heterometrous fulvipes*.

MATERIALS AND METHODS:

An experimental prospective study was designed using the viviparous animal model, *Heterometrous fulvipes*. Three groups were formed based on heavy metal exposure: group I (control), group II (mercury exposure) and group III (lead exposure). Monthly samples were drawn from different groups after exposure to mercury and lead successively at the intervals of one month. Enzymes levels were estimated in the maternal tissues (hepatopancreas, pedipalpal muscle and haemolymph) and the whole embryos in the samples drawn from group I, II and III. Samples drawn every month from August to April received one sub lethal dose per month; hence the sample from the month of August represented the effect of a single dose, whereas samples taken at September represented cumulative effect of two doses. In the same order samples at the month of April represent the effect of nine sub lethal doses of the heavy metals.

Samples of control and experimental animals were procured. 10% homogenate of muscle, 7% homogenate of liver and 2% homogenate of embryos were made in 0.025m sucrose solution at 5 degrees Celsius. Supernatants obtained after centrifugation at 3000 rpm for 15 Minutes, were used as enzyme source for assay of GDH and SDH. The protein content in the enzyme extract was estimated by the method of Lowry et. al.

Estimation of GDH:

The activity level of GDH was estimated using the method of Lee and Lardy. 2ml of reaction mixture contained 100µmoles of phosphate buffer (7.4 pH), 40 µmoles of sodium glutamate, 0.1 µmoles of NAD (Nicotinamide adenine dinucleotide), 4µmoles of the INT 2-(4-iodophenyl)-3-(4-nitrophenyl)-5-phenyl-2H-tetrazolium and 0.4 ml of homogenate. The reaction mixture was incubated at 37° C for 30 minutes and the reaction was arrested by the addition of 6 ml of glacial acetic acid. The formazan firmed overnight at 5°C was extracted in 5ml of toluene. The color developed was read at 495 µmoles of formazan formed/mg protein/h.

Estimation of SDH:

The activity levels of SDH was estimated by employing the methods at Nachlas et al. 2 ml of the reaction mixture contained 100 µmoles of phosphate buffer (7.4 pH), 40 µmoles of sodium succinate, 4 µmoles of INT and 0.5 ml of homogenate. The reaction mixture was incubated at 37°C for 30 minutes, and later the reaction was arrested by the addition of 6 ml of glacial acetic acid. The formazan formed overnight at 5° C was extracted in 5 ml of toluene. The color developed was read at 495 µmoles against a blank. The specific activity of the enzyme was expressed as micro moles of formazan formed/mg protein/h.

Colorimetric readings for all the estimations were taken using the Bausch and Lomb colorimeter (Spectronic – 20). In view of the occurrence of the marked diurnal rhythmic activity in *H. fulvipes* all the estimations were carried out between 9 a.m. and 12 p.m.

RESULTS:

Effect of mercury and lead on the activity of glutamate dehydrogenase in the maternal tissue and embryos:

A) Maternal hepatopancreas:
The activity of glutamate dehydrogenase exhibited an initial rise in September and declined in October. Thereafter the elevation of enzymatic activity was noticed in November to January and March (Table 31; Fig.40).

Administration of monthly sub lethal doses of mercury during gestational period did not alter the pattern of variation, but elevated the enzyme activity throughout the gestation period, though statistically not significant. A similar effect of lead resulting in a statistically significant elevation in the enzyme activity from the beginning to the end of gestation period was noticed.

Table-31: Effect of mercury (Hg) and lead (Pb) on the activity of levels of glutamate dehydrogenase in the hepatopancreas of *H. fulvipes* during the gestation period.

Values represent mean ± S.E. Number of observations (N) = 8.

Month of treatment	GDH µmoles of formazan formed/mg protein/hr		
	Control	Experimental	Percent elevation
AUG.	0.22 ± 0.01	Hg 0.25 ± 0.01* Pb 0.26 ± 0.01[a]	11.11 18.22
SEP.	0.25 ± 0.01	Hg 0.27 ± 0.01* Pb 0.29 ± 0.01[a]	8.59 15.23
OCT.	0.17 ± 0.01	Hg 0.19 ± 0.01* Pb 0.21 ± 0.01[a]	10.79 19.31
NOV.	0.20 ± 0.01	Hg 0.21 ± 0.01* Pb 0.24 ± 0.01[a]	8.50 21.50
DEC.	0.26 ± 0.01	Hg 0.26 ± 0.01* Pb 0.31 ± 0.01[a]	11.53 19.23
JAN.	0.28 ± 0.01	Hg 0.31 ± 0.01* Pb 0.32 ± 0.01[a]	9.89 16.25
FEB.	0.25 ± 0.01	Hg 0.28 ± 0.01* Pb 0.30 ± 0.01[a]	11.55 19.52
MAR.	0.31 ± 0.01	Hg 0.34 ± 0.01* Pb 0.36 ± 0.01[a]	8.49 11.00
APR.	0.19 ± 0.01	Hg 0.22 ± 0.01* Pb 0.24 ± 0.01[a]	13.40 23.71

$a_p < 0.05$; $b_p < 0.01$; * - insignificant

B) Pedipalpal muscle:
Enzyme activity both in controls and experimental animals steadily increased during the gestational period from August to March and declined in April (Table 32; Fig. 41).

The impact of heavy metals was reflected in the increased levels of the activity of the GDH in all cases, though statistically insignificant in mercury treated animals and significant in all but three instances in the samples treated with lead. The magnitude of effect in different months during the gestation period did not reveal any dose dependency.

Table-32: Effect of maternal treatment with mercury (Hg) and lead (Pb) on the activity levels of glutamate dehydrogenase in the pedipalpal muscle of *H. fulvipes* during the gestation period.

Values represent mean S. E. Number of observations (N) = 8.

Month of treatment	GDH µmoles of formazan formed/mg protein/hr		
	Control	Experimental	Per cent elevation
AUG.	0.13 ± 0.006	Hg 0.14 ± 0.005* Pb 0.15 ± 0.005[a]	8.14 14.81
SEP.	0.16 ± 0.006	Hg 0.18 ± 0.008* Pb 0.32 ± 0.007[a]	7.10 12.42
OCT.	0.17 ± 0.007	Hg 0.18 ± 0.10* Pb 0.20 ± 0.007[a]	7.42 17.71
NOV.	0.19 ± 0.01	Hg 0.20 ± 0.01* Pb 0.22 ± 0.01[a]	8.37 17.80
DEC.	0.19 ± 0.01	Hg 0.21 ± 0.01* Pb 0.23 ± 0.01[a]	8.04 18.59
JAN.	0.23 ± 0.01	Hg 0.24 ± 0.01* Pb 0.26 ± 0.01[a]	7.36 15.15
FEB.	0.25 ± 0.01	Hg 0.26 ± 0.01* Pb 0.28 ± 0.01[a]	7.17 13.94
MAR.	0.27 ± 0.01	Hg 0.29 ± 0.01* Pb 0.31 ± 0.01[a]	6.11 14.38
APR.	0.17 ± 0.008	Hg 0.18 ± 0.009* Pb 0.21 ± 0.01[a]	4.49 19.66

$a_p < 0.05$; $b_p < 0.01$; * - insignificant

C) Embryos:
The activity of GDH in the embryos increased continuously all through the gestational period both in the controls and experimental animals with no difference in the pattern of variation (Table 33; Fig. 42).

Administration of monthly sub lethal doses of mercury elevated the levels of activity of the enzyme, though statistically not significant, for all but the January and April. A similar effect of the lead resulting in statistically significant elevation in the enzyme activity from the beginning to the end of gestation period was noted except in March month sample.

Table-33: Effect of maternal treatment with mercury (Hg) and lead (Pb) on the activity levels of glutamate dehydrogenase in the embryos of *H. fulvipes* during the gestation period.

Values represent mean S. E. Number of observation (N) = 8.

Month of treatment	GDH µmoles of formazan formed/mg protein/hr		
	Control	Experimental	Per cent elevation
SEP.	0.53 ± 0.009	Hg 0.55 ± 0.01* Pb 0.58 ± 0.007[c]	3.54 8.76
OCT.	0.56 ± 0.01	Hg 0.58 ± 0.01* Pb 0.60 ± 0.01[a]	3.35 7.40
NOV.	0.58 ± 0.01	Hg 0.60 ± 0.01* Pb 0.63 ± 0.01[b]	3.06 7.31
DEC.	0.66 ± 0.02	Hg 0.71 ± 0.02* Pb 0.74 ± 0.03[a]	7.72 12.87
JAN.	0.83 ± 0.01	Hg 0.88 ± 0.01[b] Pb 0.93 ± 0.01[c]	6.24 12.24
FEB.	0.92 ± 0.01	Hg 0.95 ± 0.02* Pb 1.00 ± 0.03[a]	3.12 8.18
MAR.	1.10 ± 0.03	Hg 1.12 ± 0.03* Pb 1.17 ± 0.04*	2.36 6.72
APR.	1.96 ± 0.03	Hg 2.05 ± 0.03[a] Pb 2.10 ± 0.03[b]	4.57 7.07

$a_p < 0.05$; $b_p < 0.01$; $c_p < 0.001$; * - insignificant

Effect of mercury and lead on the activity levels of SDH in maternal tissues and embryos:

A. Maternal hepatopancreas:
The sub lethal doses of the heavy metals, mercury and lead administered to the gravid females every month from August to April, brought about depressions in the SDH activity at all points, without any deviation from the controls in the patterns of variation during the gestation period (Table 34; Fig. 43). It could however, be noted that the effect of lead was statistically significant during the later part of gestational period i.e., from January onwards.

Table-34: Effect of mercury (Hg) and lead (Pb) on the activity levels of succinic dehydrogenase in the hepatopancreas of *H. fulvipes* during the gestation period.

Values represent mean ± S. E. Number of observations (N) = 8.

Month of treatment	SDH µmoles of formazan formed/mg protein/hr		
	Control	Experimental	Per cent elevation
AUG.	0.423 ± 0.012	Hg 0.368 ± 0.015[b] Pb 0.348 ± 0.012[c]	13.002 17.730
SEP.	0.456 ± 0.016	Hg 0.420 ± 0.014[a] Pb 0.396 ± 0.012[a]	7.895 13.158
OCT.	0.487 ± 0.013	Hg 0.442 ± 0.014[a] Pb 0.423 ± 0.014[b]	9.240 13.141
NOV.	0.507 ± 0.016	Hg 0.467 ± 0.016[a] Pb 0.449 ± 0.013[b]	7.890 11.440
DEC.	0.428 ± 0.011	Hg 0.400 ± 0.010[a] Pb 0.384 ± 0.014[a]	6.543 10.280
JAN.	0.450 ± 0.014	Hg 0.418 ± 0.012* Pb 0.401 ± 0.012[a]	7.112 10.889
FEB.	0.406 ± 0.010	Hg 0.376 ± 0.016* Pb 0.348 ± 0.013[b]	7.17 13.94
MAR.	0.440 ± 0.010	Hg 0.413 ± 0.010* Pb 0.385 ± 0.015[b]	6.11 14.38
APR.	0.371 ± 0.011	Hg 0.355 ± 0.010* Pb 0.340 ± 0.010[a]	4.49 8.356

$ap < 0.05$; $bp < 0.01$; * - insignificant

B) Pedipalpal muscle:
The sub lethal doses of mercury and lead exerted a depressant action on the succinic dehydrogenase activity in the pedipalpal muscle of the maternal animal

without any deviation from variation in the activity levels of SDH during different months of gestational period (Table 35; Fig. 44). The effect was statistically significant with lead but not with mercury. The per cent depletion in the sample of every month showed that the effect was not reflecting any dose dependency.

Table-35: Effect of mercury (Hg) and lead (Pb) on the activity levels of succinic dehydrogenase in the pedipalpal muscle of *H. fulvipes* during the gestation period.

Values represent mean S. E. Number of observations (N) = 8.

Month of treatment	SDH µmoles of formazan formed/mg protein/hr		
	Control	Experimental	Per cent elevation
AUG.	0.280 ± 0.008	Hg 0.258 ± 0.010[a] Pb 0.245 ± 0.008[b]	7.858 12.500
SEP.	0.335 ± 0.010	Hg 0.317 ± 0.011[*] Pb 0.300 ± 0.010[a]	5.373 8.060
OCT.	0.301 ± 0.010	Hg 0.283 ± 0.013[*] Pb 0.260 ± 0.012[a]	5.981 13.622
NOV.	0.366 ± 0.012	Hg 0.340 ± 0.012[*] Pb 0.321 ± 0.011[b]	7.104 12.295
DEC.	0.334 ± 0.011	Hg 0.310 ± 0.010[*] Pb 0.294 ± 0.009[b]	7.186 11.977
JAN.	0.285 ± 0.010	Hg 0.270 ± 0.010* Pb 0.253 ± 0.013[a]	5.264 11.229
FEB.	0.309 ± 0.010	Hg 0.288 ± 0.011* Pb 0.274 ± 0.009[a]	6.797 11.327
MAR.	0.339 ± 0.012	Hg 0.318 ± 0.010* Pb 0.300 ± 0.010[a]	6.195 11.505
APR.	0.215 ± 0.011	Hg 0.230 ± 0.010* Pb 0.210 ± 0.010[b]	6.375 11.554

ap < 0.05; bp < 0.01; * - insignificant

C) Embryos:

The impact of administration of sublethal doses of mercury and lead to the maternal animal altered the levels of the SDH activity in the embryos leading to its decrement (Table 36; Fig. 45). The effect was statistically significant with lead but not with mercury. The magnitude of inhibition of enzyme activity by the heavy metals did not reveal dose dependent impact.

Table-36: Effect of maternal treatment with mercury (Hg) and lead (Pb) on the activity levels of succinic dehydrogenase in the embryos of *H. Fulvipes* during the gestation period.

Values represent mean S. E. Number of observations (N) = 8.

Month of treatment	SDH µmoles of formazan formed/mg protein/hr		
	Control	Experimental	Per cent elevation
SEP.	0.554 ± 0.016	Hg 0.525 ± 0.015[*] Pb 0.513 ± 0.013[a]	5.235 7.400
OCT.	0.570 ± 0.018	Hg 0.550 ± 0.017[*] Pb 0.528 ± 0.014[a]	3.509 7.369
NOV.	0.590 ± 0.019	Hg 0.573 ± 0.018[*] Pb 0.551 ± 0.017[*]	2.882 6.610
DEC.	0.711 ± 0.018	Hg 0.684 ± 0.017[*] Pb 0.652 ± 0.021[a]	3.798 8.299
JAN.	0.763 ± 0.013	Hg 0.725 ± 0.022[*] Pb 0.693 ± 0.022[b]	4.981 9.175
FEB.	0.826 ± 0.020	Hg 0.802 ± 0.020* Pb 0.766 ± 0.013[a]	2.906 7.264
MAR.	0.926 ± 0.018	Hg 0.902 ± 0.014* Pb 0.890 ± 0.015[a]	2.592 6.048
APR.	0.970 ± 0.022	Hg 0.938 ± 0.012* Pb 0.907 ± 0.015[a]	3.299 6.495

ap < 0.05; bp < 0.01; * - insignificant

DISCUSSION:

Stress due to heavy metals is known to elevate the levels of glutamate dehydrogenase and aspartate amino transferase. There are reported cases of the increase in the activity of GDH in the intestine, gills and muscles of *Channa punctatus* following exposure to mercury chloride and elevation in the activity levels of alanine aminotransferase (AAT) in the kidney, brain and the liver tissue of Sprague-Dawley rats following exposure to cadmium (9, 13). The depletion of proteins under the stress of the heavy metals, mercury and lead observed in the maternal tissues of *H. fulvipes,* indicates proteolysis, suggesting that the proteins are utilized for meeting the excess energy demand imposed by the toxic stress.

Elevation in the levels of GDH and AAT in the maternal tissue of *H. fulvipes* can be considered a response to the stress induced by the heavy metals, mercury and lead to generate keto acids like α – ketoglutarate and oxaloacetate for contributing to gluconeogenesis and/or energy production necessary to meet the excess energy demand under the toxic manifestations. GDH catalzses the reversible deamination of glutamate to - ketoglutarate and ammonia. Thus, the amino transferases with GDH contribute some strategic substances such as - ketoglutarate, pyruvate, oxalo-acetate and glutamate for various synthetic and oxidative reactions. The elevation of AAT activity provides the oxaloactic acid required for the gluconeogenic pathway, to meet the additional supply of glucose for the production of energy under reduced phase of oxidative metabolism. The alterations in the levels of activity of glutamate dehydrogenase and aminotransferases by the heavy metals, mercury and lead, clearly indicate that the stress brings about the metabolic reorientations in the maternal tissues by raising energy resources GDH and transaminase system. The inhibition of the activity of alanine aminotransferase in the maternal tissue of *H. fulvipes* under the heavy metal treatment, in contrast to GDH and AAT, is suggestive of specific impact of mercury and lead in different enzymes system.

Succinic dehydrogenase is an important enzyme in oxidative metabolism. Treatment with sublethal doses of mercury and lead depressed the activity levels of SDH in the maternal tissue of *H. fulvipes*. The decline is indicative of the depressant action of the heavy metals on the oxidative metabolism. Similar depressant action of mercury on the levels of activity of succinic dehydrogenase in the brain, gills, intestine, kidney, liver and muscles of Channa punctatus are demonstrated earlier (4). The depletion of the SDH in the present study is probably met by augmented rate of anaerobic oxidation, which is reflection of the reduction of excess energy under the toxic manifestations of the heavy metals. Enhanced demands for glucose under such circumstances are probably met by gluconeogenesis from proteins mediated by GDH and transaminases.

The responses of the enzyme system in the embryos of the maternal animals treated with sublethal doses of mercury and lead are basically similar to, and not much different from, the responses observed in the maternal tissues. This probably suggest that the embryo behaves as an integral part of the maternal tissues and cannot be considered to reflect any autonomous status. The reduction in the size of embryo, observed when the maternal animal was treated with the heavy metals during the gestation period, can hence be attributed to the excessive utilization of the biochemical constituents for energy needs, mediated by their enzyme systems, both by the mother and embryos, apart from the consequents lowered supplies of nutrients from the mother to the embryos.

Disclaimer: None

Conflict of Interest: None

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FIGURES:

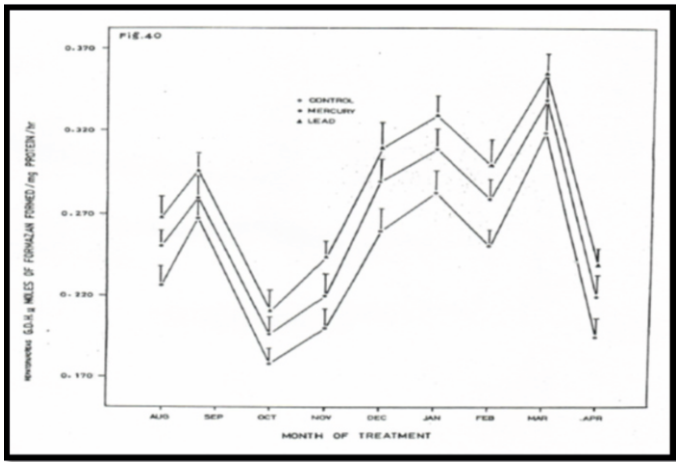

Fig-40: Effect of mercury and lead on the activity levels of glutamate dehydrogenase in the hepatopancreas of *H. fulvipes* during the gestation period.

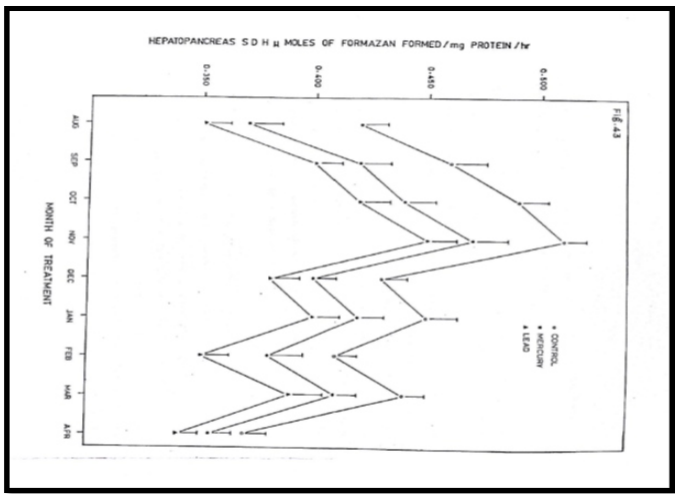

Fig-43: Effect of mercury and lead on the activity levels of succinic dehydrogenase in the hepatopancreas of *H. fulvipes* during the gestation period.

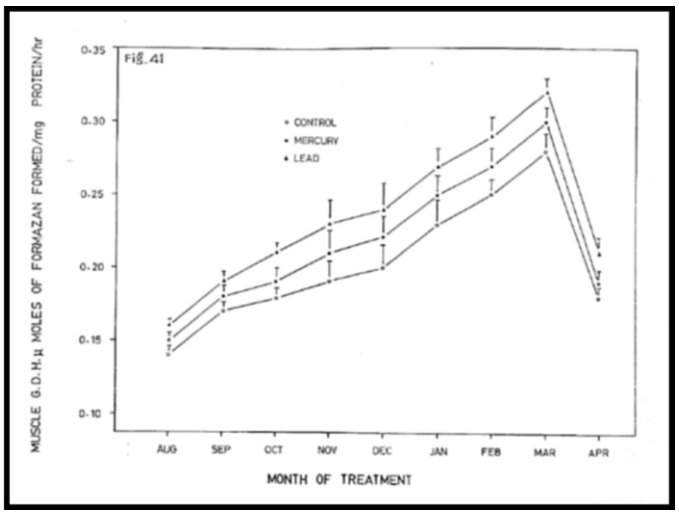

Fig-41: Effect of mercury and lead on the activity levels of glutamate dehydrogenase in the pedipalpal muscle of *H. fulvipes* during the gestation period.

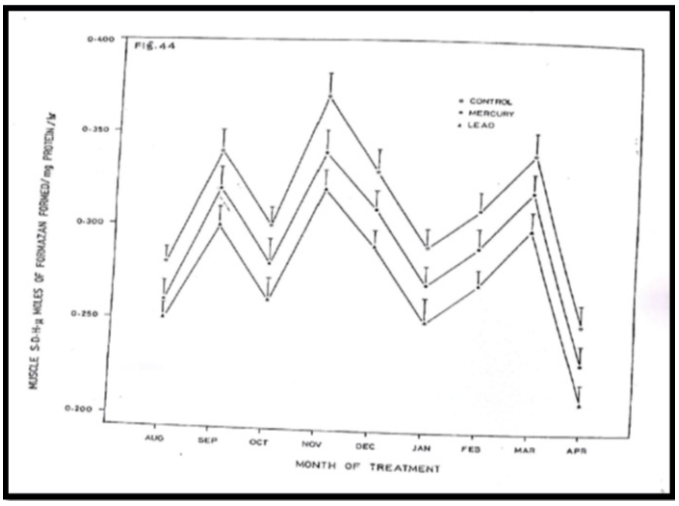

Fig-44: Effect of mercury and lead on the activity levels of succinic dehydrogenase in the pedipalpal muscle of *H. fulvipes* during the gestation period.

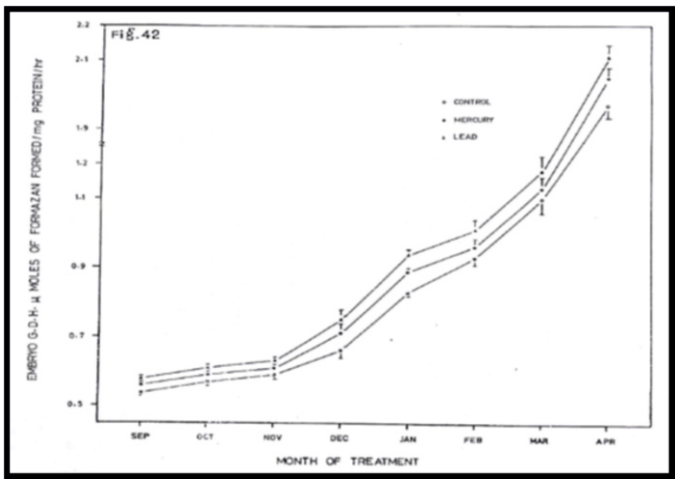

Fig-42: Effect of maternal treatment with mercury and lead on the activity levels of glutamate dehydrogenase in the embryos of *H. fulvipes* during the gestation period.

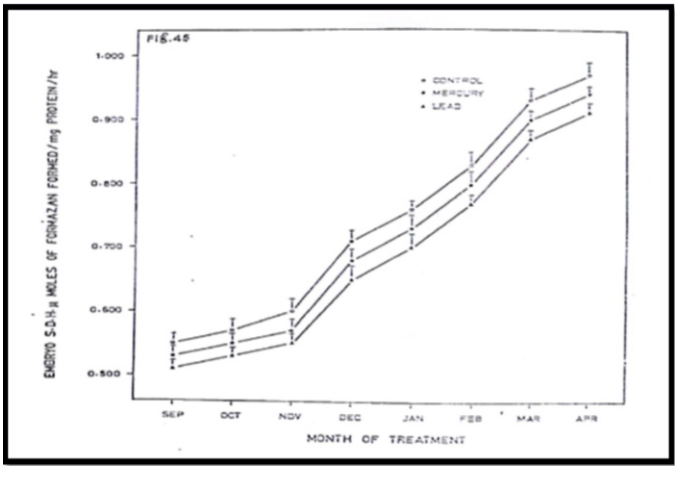

Fig-45: Effect of maternal treatment with mercury and lead on the activity levels of succinic dehydrogenase in the embryos of *H. fulvipes* during the gestation period.